

SLEEP DEPRIVATION AND EXERCISE TOLERANCE(U) INDIANA
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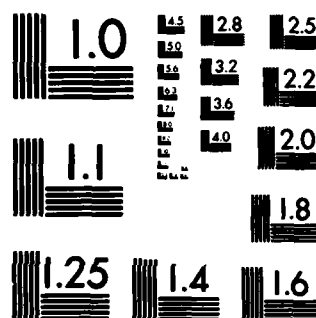
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REPORT NUMBER 4

Sleep Deprivation and Exercise Tolerance

Annual Summary Report

Bruce J. Martin, Ph.D.

January 1985

Supported by

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND

Fort Detrick, Frederick, Maryland 21701-5012/

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Indiana University School of Medicine

Bloomington, Indiana 47405

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number) The purpose of the study is to identify the effects of sleep deprivation on the physiological and psychological responses to exercise. Standard techniques in human exercise physiology are utilized. During this year, we found that fragmenting two nights of sleep prior to heavy exercise had no effect on physiological response to that exercise. (CONTINUED ON BACK)		

Heart rate, metabolic rate, and body temperature were identical to control, in contrast to sleepiness and mood disturbance, both of which were significantly elevated. In past work, such psychological effects were correlated with decreased exercise tolerance. Also, we found that a 36 hour sleepless period prior to prolonged mild exercise had no influence on physiological responses to that exercise, though it as well significantly elevated mood disturbance. Because neither form of sleep loss changed stress hormonal levels in subsequent exercise, we conclude that sleep loss of this form is primarily a psychological and not a physiological stress.

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SUMMARY

The purpose of the study is to identify the effects of sleep deprivation on the ability of humans to tolerate standard forms of endurance exercise. Standard techniques in human exercise physiology are utilized.

To date, our results show a) that a 30 hour sleep loss period does not reduce $\dot{V}O_{2\max}$, or short-term exercise tolerance, while it leads to a 10% decline in the ability to carry out prolonged exhausting exercise, b) that a 50 hour sleepless period reduces long-term exercise performance about 20%, independent of any clear-cut physiological changes, c) that exercise in severe environmental conditions (cold) is unaffected by a 50 hour sleepless period, and d) that sleep loss does not alter the sympathoadrenal or other stress hormonal responses to subsequent exercise.

We conclude that sleep loss has cumulative effects upon subsequent exercise performance, with these effects being mediated through psychological, not physiological effects.

FOREWARD

For the protection of human subjects the investigator(s) have adhered to policies of applicable Federal Law 45CFR46.

BODY OF REPORT

STATEMENT OF THE PROBLEM

Sleep deprivation is a common occurrence in both the military and civilian spheres. In many instances, severe or prolonged exercise follows sleep loss. Despite this frequent occurrence, little is known of the effects that sleep deprivation may have on subsequent exercise.

BACKGROUND

Several studies have now shown a decline in prolonged exercise tolerance after sleep deprivation. Two of them have taken place in earlier years under this contract. In the first, a 36 hour sleepless period decreased subsequent prolonged exercise tolerance by about 10%, a change that was also statistically significant ($p < 0.05$) (1). Individual responses varied widely in this study, with half of subjects showing essentially no effect, while the other 50% exhibited declines of at least 15%, ranging up to 40%. This result reveals how individual susceptibility to sleep loss varies within a typical population sample. In a subsequent study (2), a 50 hour sleepless period preceded similar, prolonged heavy exercise to exhaustion, and created a 20% deficit in performance, a decrease that was statistically significant ($p < 0.01$) and almost significantly greater ($0.05 < p < 0.10$) than the performance decrement produced by a 36 hour sleep loss. In neither study were there physiological changes that could explain the performance changes: oxygen uptake, minute ventilation, heart rate, respiratory quotient, and blood lactic acid, norepinephrine, epinephrine, and dopamine concentrations were all unaffected by sleeplessness. Because sleep loss did produce a measurable disturbance of mood, we tentatively concluded that psychological, not physiological, mechanisms underlay the performance decrement.

In a further study that tended to confirm this hypothesis, we used severe cold stress as a motivation for heavy exercise. In this study (3), we found that a 50 hour sleepless period did not change this work load selected for thermal and exercise fatigue comfort, nor did it change the duration for which subjects were willing to endure cold exposure.

APPROACH TO THE PROBLEM

These past studies led us to more carefully consider the mechanism by which sleep loss influences exercise tolerance. If, indeed, these mechanisms are strictly psychological in nature (and thus have, as yet, no well-defined physiological correlates), then sleep loss *per se* is not, after all (4, 5), a classic physiological stress, and further, full arousal by whatever available means should minimize or even eliminate any exercise performance decrement induced by sleeplessness. We thus investigated whether or not sleep deprivation alters the stress hormonal response to exercise.

In a first study (described in a previous annual report (6)), we used two nights of fragmented sleep as the sleep deprivation protocol. In a further study, performed in the past year, a 36 hour sleepless period preceded long-term exercise. These two studies are reported together below as their results, similar qualitatively, lead to the same conclusions.

RESULTS AND DISCUSSION

Briefly, in review, we found that two nights of fragmented sleep failed to change any physiological responses to subsequent heavy exercise at 70% $\dot{V}O_{2max}$. These included heart rate, metabolic rate, body core temperature, or ventilation, and, as well, stress hormones: norepinephrine, epinephrine, cortisol, and β -endorphin. The hormonal levels were measured after 30 min exercise. In contrast, sleepiness (as assessed by the Stanford Sleepiness Scale (7)), and mood disturbance (quantified by the Profile of Mood State (POMS; 8)) were both significantly increased prior to exercise ($p < 0.05$). Exercise itself eliminated the sleepiness increase seen after sleep loss, but it did not mitigate the mood disturbance. Thus, as before, psychological and not physiological response to exercise were altered by sleep deprivation.

Our most recent study used a different sleep loss duration (36 consecutive hours) and different exercise (3 hours of walking at $5.6 \text{ km} \cdot \text{hr}^{-1}$ up a 2% grade, a work rate requiring use of about 25% of the $\dot{V}O_{2max}$) to examine the same questions asked earlier, from a different perspective. As before, we found no influence of sleeplessness on heart rate, oxygen uptake, body core temperature, or minute ventilation, while mood disturbance and sleepiness were significantly increased ($p < 0.05$) prior to exercise. In parallel with the earlier study, mood remained disturbed throughout exercise, while sleepiness was mitigated after 2 hours of work such that it was equal to control levels. Finally, as in the previous study, we found no evidence to suggest that sleep loss alters the stress hormonal response to exercise. Rather, blood cortisol, catecholamines, and β -endorphin were identical in the two situations.

CONCLUSIONS

Sleep loss decreases tolerance of heavy endurance exercise, possibly through psychological alterations manifest as decreased arousal. Sleep loss per se has no apparent physiological effects in subsequent exercise, and is not a physiological stress.

RECOMMENDATIONS

Sleep loss is relatively ineffective as a direct inhibitor of exercise tolerance, once the need and motivation for such exercise is established. Its potent effects are psychological, with mood disturbance (toward greater fatigue and confusion) and presumably decreased self-motivation being the primary deficits that influence both the ability to tolerate, and the desire to initiate, exercise.

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